Clinical Guidance

Paediatric Critical Care: Acid Base Interpretation

Summary

This guideline is for the use by clinical staff who are interpreting acid base balance in critical care. Rather than focusing on bicarbonate which is a derived value, interpretation is aided by accounting for the contribution made to acid-base by: chloride, albumin and unmeasured acids.

This clinical guideline has been produced by the South Thames Retrieval Service (STRS) at Evelina London for nurses, doctors and ambulance staff to refer to in the emergency care of critically ill children.

This guideline represents the views of STRS and was produced after careful consideration of available evidence in conjunction with clinical expertise and experience. The guidance does not override the individual responsibility of healthcare professionals to make decisions appropriate to the circumstances of the individual patient.
Acid base interpretation

- Traditional teaching relies on the Henderson-Hasselbalch formula that states \( \text{HCO}_3^- \) (metabolic component) and \( \text{pCO}_2 \) (respiratory component) can vary independently according to the formula \( \text{pH} = 6.1 + (\text{HCO}_3^-/\text{pCO}_2 \text{ mmHg} \times 0.003) \)
- This assumes bicarbonate is the major and only significant buffer for acidosis.
- In clinical practice the Base Excess is used to “quantify” the magnitude of metabolic component of an acid base derangement. Bicarbonate and base excess are not directly measured (they are calculated from pH and \( \text{pCO}_2 \) so they are always coupled and dependent on these. For this reason they can’t be used as direct measures of acid base.
- Both bicarbonate and base excess do not correlate well in-vivo with acid base disturbance because they assume all other blood components and electrolytes are normal (Hb, Na, Cl, Albumin) which is rare in sick patients as electrolyte problems are common.
- Stewart’s strong ion methodology explains pH changes for all three major buffer systems: 1) carbonic acid (\( \text{pCO}_2 - \text{bicarbonate} \)), 2) Electrolytes (Na, K, Cl, Lactate, Ca, Mg) and 3) weak acids (albumin and phosphate). Although complex, it can be simplified at the bedside to explain acid base disturbances where electrolyte abnormalities are present and is hence more accurate.

In Stewart’s model, the bicarbonate space has to compete with other negatively charged anions in order to maintain electroneutrality:

Anion charge = Cation charge

or

\[ \text{Na}^+ + \text{K}^+ + \text{Ca}^{2+} + \text{Mg}^{2+} = \text{Cl}^- + \text{HCO}_3^- + \text{lactate} + \text{albumin} \text{ charge}. \]

1. If Cl is very high, bicarb is squeezed into a smaller space (hyperchloraeic acidosis) Figure 1b
2. If Cl is very low, bicarb has more space to occupy (hypochloraeic alkalosis) Figure 1c
3. If albumin is low, it will allow more space for bicarbonate (low albumin is alkalizing) Figure 1d
4. If albumin is high, it will reduce the space bicarbonate has (high albumin is acidifying)
5. If an anionic acid is present like lactate or ketones, a low Cl (Figure 1e) or in combination with a low albumin (Figure 1f) may partially buffer acidosis by allowing more space for bicarb to be in. This is common in clinical practice
ROLE OF CHLORIDE

1. Chloride to sodium ratio

Simplistically, Cl must always be interpreted relative to Na. The chloride to sodium ratio (Cl:Na) is easiest way to do this:

Normal Cl = 106, normal Na = 140 so Cl:Na = 0.75 or Cl is 75% of Na (range 72-80%)
- Cl is frankly acidifying if Cl > 80% of Na
- Cl is frankly alkalinizing if Cl < 72% of Na

All these cases demonstrate hyperchlaema as an acidifying force regardless of absolute chloride (it’s the relationship with Na that’s important) Cl 115 and Na 140 or Cl 107 and Na 130 or Cl 135 and Na 160.

(DO NOT CORRECT CHLORIDE FOR BODY WATER: it gives false values because Cl and Na do not distribute equally between ICF and ECF)

2. Partitioning base deficit for chloride and sodium (BEcl)

The effect of chloride and sodium on the base excess can be simplified to the following formula:

Base excess due to chloride and sodium = Na – Cl – 32

If Base excess = -10 mEq/L, Na = 140, Cl = 113, then chloride accounts for 140 – 113 -32 = - 5 mEq/L

I.e. Chloride is acidifying by 5 mEq/L or 50% of the base excess (the remaining 5mEq/L could be explained by other anionic acids (e.g. lactate, acids broken down from glycocalyxy in sepsis)

EFFECT OF ALBUMIN

- Albumin is a weak acid and has a charge in mEq/L of about 25% of the concentration in g/L (i.e. 40g/L has a charge of about 10mEq/L). The effect of albumin can be simplified by the following formula:

Albumin effect on base excess = (42 – Albumin g/L) x 0.25 = + mEq/L

For example, if albumin is low at 32 g/L, the base excess will be increased by the alkalinizing effect of low albumin by:

Albumin effect base excess: (42 – Albumin g/L) x 0.25 = 42 – 32 x 0.25 = + 2.5 mEq/L (low albumin is therefore alkalinizing)

- Rule of thumb: Every 10 g/L fall of albumin will increase the base excess by 2.5 mEq/L.

- The anion gap is also falsely lowered by low albumin. To correct the anion gap for low albumin:

Corrected anion gap = anion gap + (42 – Albumin g/L) x 0.25

UNMEASURED ANIONS

- Acids are usually present as acidifying anions (e.g. lactate or ketones). As they are not routinely measured, their presence can be inferred by a raised anion gap (>16 where AG is Na + K – Cl – Bic) once corrected for albumin

- Alternatively the effect of “unmeasured anions” can be quantified by the partitioned base excess by the difference between all known components of base excess (albumin and chloride) form the total base excess:

Base ExcessUNMEASURED = SBE – BECHLORIDE – BEALBUMIN

- Because Cl falls as a compensatory phenomenon, a low Cl:Na ratio with acidosis (<0.74) usually indicates the presence of large amounts of unmeasured acids. Conversely a very high Cl:Na ratio > 0.85 excludes other acids like lactate as cause of an acidosis. When hyperchlaema co-exists with other acids like lactate the Cl:Na ratio is usually in between 0.74 and 0.80.

FULL STEWART EQUATIONS

The full Stewart equations can be used accurately quantify all the above components. They are not provided here as they are complex and require multiple calculations. The above formulas are simple bedside approximations
ACID BASE MADE SIMPLE: CLINICAL APPLICATION

1. Define nature of disturbance (low pH = acidaemia, high pH = alkalaemia)
2. Define what forces are acidifying and which are alkalinizing and how they balance:

<table>
<thead>
<tr>
<th>Acidifying force</th>
<th>Alkalinizing force</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory component</td>
<td>High pCO₂</td>
</tr>
<tr>
<td>Chloride</td>
<td>hyperchloreaemia</td>
</tr>
<tr>
<td>Albumin</td>
<td>hyperalbuminaemia</td>
</tr>
<tr>
<td>Phosphate*</td>
<td>hypophosphataemia</td>
</tr>
</tbody>
</table>

* Usually values too small to influence acid base in major way therefore not included

FORMULAS:

- **Cl:Na**: 
ger > 80% = frank hyperchloreaemia, <72% = frank hypocholeaemia  
- **Base excess Cl**: 
  \[ \text{Base excess Cl} = \text{Na} - \text{Cl} - 32 \]  
- **Base excess Albumin**: 
  \[ \text{Base excess Albumin} = (42 - \text{Albumin g/L}) \times 0.25 \]  
- **Base excess unmeasured anions**: 
  \[ \text{Base excess (total)} - \text{BE} \text{Cl} - \text{BE} \text{ALB} \]

Clinical Example: Blood gas: pH 7.0 pCO₂ 3.8 kPa, Standard Bicarb 10 SBE -10mEq/L Na 125, Cl 107, Alb 22 g/L

**STEP 1. WHAT IS THE pH?**
pH = very low = ACIDOSIS (not respiratory as pCO₂ is low)

**STEP 2. WHAT ARE ACIDIFYING AND ALKALISING PROCESSES?**

- **Cl:Na**: 
  \[ \text{Cl:Na} = 0.86 = 86\% = \text{frankly acidifying, BE} \text{Cl} = \text{Na} - \text{Cl} - 32 = 125 - 107 - 32 = -14 \text{ mEq/L} = \text{acidifying} \]
- **BE ALBUMIN**: 
  \[ \text{BE ALBUMIN} = (42 - \text{Alb}) \times 0.25 = +5 \text{ mEq/L} = \text{alkalizing} \]
- **BE UNMEASURED**: 
  \[ \text{BE UNMEASURED} = -10 - -14 - 5 = -1 \text{ mEq/L}, \text{ Anion Gap} = 12 \text{ (corrected for albumin = 17)} \]

<table>
<thead>
<tr>
<th>Acidifying</th>
<th>Alkalinizing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>Low pCO₂ respiratory compensation</td>
</tr>
</tbody>
</table>
| Chloride | Hyperchloreaemia +++  
  Cl:Na >80%, BECl -14mEq/L |
| Albumin | Hypoalbuminaemia  
  BE ALBUMIN = +5mEq/L |
| Unmeasured | BE UNMEASURED = -1  
  AG corrected = 17 |

Interpretation

1. Severe acidosis (metabolic)
2. Some respiratory compensation
3. Main cause acidosis = severe hyperchloreaemia
4. Minimal degree "tissue" acidosis as anion gap and unmeasured anions only slightly raised
5. Albumin is very low and alkalinising by a significant amount (5 mEq/L of BE)

NB: It is the net combination of acidifying and alkalinizing forces that determine pH

2) O'Dell Crit Care. 2005 vol 9:R464-70